UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF MASSACHUSETTS

JOHN HANCOCK LIFE INSURANCE COMPANY, JOHN HANCOCK VARIABLE LIFE INSURANCE COMPANY, and MANULIFE INSURANCE COMPANY (f/k/a INVESTORS PARTNER INSURANCE COMPANY), Plaintiffs, vs. ABBOTT LABORATORIES, Defendant.	Civil Action No. 05-11150-DPW Hon. Judge Douglas P. Woodlock

CORRECTIONS TO THE AFFIDAVIT OF JOHN MARTIN LEONARD, M.D.

Abbott Laboratories ("Abbott") respectfully submits these corrections to the Affidavit of John Martin Leonard, M.D. The corrections are to paragraphs 12, 43, 65, and 66 of Dr. Leonard's affidavit. The affidavit with these corrections is attached hereto as Exhibit A. Abbott will provide the court with courtesy copies of Dr. Leonard's affidavit with these corrections on Monday, March 10, 2008.

Dated: March 8, 2008

Respectfully submitted,

ABBOTT LABORATORIES

By its attorneys

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CERTIFICATE OF SERVICE

I hereby certify that this document(s) filed through the ECF system will be sent electronically to the registered participants as identified on the Notice of Electronic Filing (NEF) and paper copies will be sent to those indicated as non registered participants on March 8, 2008.

Date: March 8, 2008.

/s/ Ozge Guzelsu

Exhibit A

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MASSACHUSETTS

JOHN HANCOCK LIFE INSURANCE COMPANY, JOHN HANCOCK VARIABLE LIFE INSURANCE COMPANY and MANULIFE INSURANCE COMPANY, Plaintiffs, v. ABBOTT LABORATORIES, Defendant.	CIVIL ACTION NO. 05-11150-DPW

AFFIDAVIT OF JOHN MARTIN LEONARD, M.D.

I, John Martin Leonard, M.D., hereby declare and say:

1. My name is John Leonard. I am over 18 years of age, and suffer from no condition or disability that would impair my ability to give sworn testimony. This affidavit is based upon my own personal knowledge.

Educational and Professional Background

2. I am currently employed at Abbott Laboratories ("Abbott") as the Senior Vice-President of Global Pharmaceutical Research and Development.

3. I attended the University of Wisconsin and graduated in 1979 with a Bachelor of Arts degree in Biochemistry. I attended The John Hopkins University School of Medicine and graduated with an M.D. in 1983. I completed my medical internship and residency at Stanford University Hospital from 1983 to 1986. From 1986

to 1989 I was a postgraduate fellow in the National Institute of Allergy and Infectious Diseases at the National Institutes of Health (NIH). After I left the NIH in 1989, I briefly worked at G.H. Besselaar Associates, a contract research organization that conducted clinical trials.

4. I began working at Abbott in March 1992 as the Venture Head of the Anti-Viral Venture. In 1996, I became the Divisional Vice-President for anti-infective disease. I was promoted to Divisional Vice-President of Ventures in 1997 and, in 1999, I became the Corporate Vice-President for Development of Abbott's Pharmaceutical Products Division. In that position, I was responsible for the pharmaceutical development activities of that division. More specifically, I was responsible for supervising clinical work relating to oncology, neuroscience and anti-infective compounds under development. ABT-518, ABT-594, ABT-773 and ABT-492, among many other compounds, were within my area of responsibility. My responsibilities also included supervising some non-clinical activities, such as formulation of compounds, pre-clinical animal work as well as the statistics and data management group. In 2001, I became Corporate Vice-President for Global Pharmaceutical Development, also in the Pharmaceutical Products Division. Jeffrey Leiden, who joined Abbott in late 2000, was my immediate supervisor. In my various positions at Abbott, I have been responsible for conducting and/or supervising a substantial number of clinical trials on behalf of the company.

5. In 2004, I became the Corporate Vice-President of Global Medical and Scientific Affairs. I was promoted to Corporate Vice-President of Global Pharmaceutical Research and Development in April 2006, and recently promoted to Senior Vice-

President of Global Pharmaceutical Research and Development. In these positions, I headed and continue to head Abbott's Pharmaceutical Research and Development organization.

Abbott's Research and Development Process for New Therapeutic Drugs

6. Abbott is a global healthcare company. Its principal business is the discovery, development, manufacture and sale of a broad line of health-care products, including pharmaceuticals. Abbott's total expenditures for research & development for 2000 and 2001 were approximately $1.246 billion and $1.492 billion, respectively. Research and development expenditures have increased each year and currently exceed $2.2 billion.

7. At Abbott, as with other major pharmaceutical companies, new therapeutic drugs in development typically go through three "phases" of clinical testing in humans after passing out of the discovery and pre-clinical phases. The discovery and pre-clinical phases of pharmaceutical research and development involve the identification of molecules that are candidates for clinical testing, their characterization in *in vitro* assays, both toxicological and metabolic testing, as well as the creation of a formulation suitable for subsequent human testing of the active ingredient. In Phase I of the clinical development process, testing is typically done on a relatively small number of human volunteers, who are generally healthy. The principal purpose of Phase I testing is gain early information on the safety and toxicity of compounds along with pharmacokinetic information to permit the selection of doses appropriate for testing in patients. In Phase II, the compounds are administered to patients afflicted with the condition that the drug is intended to treat. Unlike the trials in Phase I, Phase II trials are larger and typically

attempt to determine the efficacy in addition to the safety of a variety of doses. Phase II trials include dose-ranging trials to establish the doses that will be tested in subsequent pivotal trials in patients; additional dose-ranging work is often done in Phase III clinical trials. During Phase III, the last phase of clinical development done before Abbott, like other drug sponsors, seeks regulatory approval for the new drug, the size of the clinical trials is significantly increased and the focus of the research is to confirm the efficacy and safety of the drug at the intended final dose or doses in the final patient population. If the new drug passes successfully through the three principal phases of clinical development, Abbott's clinical research and regulatory affairs teams will prepare a New Drug Application ("NDA") for the FDA's review. If the FDA approves the NDA, the new drug may be brought to market in the United States.

Negotiation of the RFA and Creation of the Descriptive Memoranda

8. I was informed in early 2000, in my role as Corporate Vice-President for Development that Abbott Laboratories was negotiating a contract with John Hancock Life Insurance Company ("Hancock") for the purpose of acquiring additional funding to share the cost of developing several of Abbott's key pre-clinical and clinical compounds. I was enthusiastic about partnering with Hancock because I believed that the shared development strategy would be advantageous for both Abbott and our future partner.

9. I was not directly involved in the negotiation of the terms of the research funding agreement ("RFA") between Abbott and Hancock. I am aware that the RFA provided that Hancock would contribute funding for nine pharmaceutical compounds (the "Program Compounds") including ABT-518, ABT-594, and ABT-773. As discussed above, as Corporate Vice-President of Development for Abbott's Pharmaceutical

Products Division, I was responsible for the development of these three compounds in 2000, and in 2001 when I was promoted to Corporate Vice-President for Global Pharmaceutical Development. I was aware at that time of the general development status and prospects for ABT-518, ABT-594, and ABT-773.

10. In July 2000, before the RFA was executed, I participated in a telephone call with Mr. Stephen Cohen of Abbott, Mr. Stephen Blewitt of Hancock and Dr. Lynn Klotz, who I understood was an independent scientific consultant retained by Hancock to assist Hancock's due diligence with regard to the compounds. During this call, I answered several questions posed by Dr. Klotz and Mr. Blewitt regarding the development status of many of the Program Compounds. Most of the questions were posed to me by Dr. Klotz. I attempted to answer all of the questions, based on my personal knowledge regarding the Program Compounds. Although I do not recall the specifics of the discussions, I have a general recollection that we discussed the side effect profile of ABT-594. At that time I understood that the dose limiting side effects were not dangerous, like hypothermia and seizures, but less severe, including headaches and vomiting. As discussed below, these side effects were also disclosed in the versions of the descriptive memoranda we gave to John Hancock.

11. The RFA included Descriptive Memoranda that were included as exhibits to the RFA. They were drafted at the direction of Steve Cohen, the controller of the Pharmaceutical Products Division Research and Development Group. I did not draft the Descriptive Memoranda that were included with the RFA. They were drafted by individuals in New Product Development and the respective heads of the teams developing the compounds that are the subject of the memoranda. Members of the

development teams for each of the various compounds were primarily responsible for reviewing and modifying the Descriptive Memorandum for their respective compounds.

12. I reviewed earlier drafts of some of the Descriptive Memoranda and Annual Research Plans, including the November 2000 Descriptive Memoranda. Attached hereto as D's Exhibit A are true and correct copies of the November 2000 Descriptive Memoranda and Annual Research Plans for ABT-518, ABT-594, and ABT-773 with my handwritten notes on the documents. As set forth in my notes, after reviewing the draft Descriptive Memoranda and Annual Research Plans, I concluded that they were well written and would provide Hancock with the information that it wanted. *Id.* at ABBT0006628. I reviewed some of the final February 2001 Descriptive Memoranda before the RFA was executed, including the final Descriptive Memorandum for ABT-518. I cannot recall whether I reviewed the final Descriptive Memoranda for all of the compounds.

13. My purpose in reviewing the Descriptive Memoranda was to confirm the accuracy of the information contained within them. If I determined, based on information that I had received regarding the Program Compounds, that anything included in the draft Descriptive Memoranda that I reviewed was inaccurate, I either reported the inaccuracy to the individual responsible for drafting the Descriptive Memoranda in order to have it corrected or I annotated the document itself for correction. For example, I noticed with regard to the Descriptive Memorandum for ABT-518 that the draft Descriptive Memorandum stated that the Phase I clinical trial for ABT-518 started in December 2000. By the time I reviewed this draft memorandum, the beginning of this

trial had actually been pushed back to March 2001. Accordingly, I noted the need to correct this information in the Descriptive Memorandum before the RFA was executed.

14. I am aware that ABT-980 was one of the compounds that was originally planned to be included as a Program Compound in 2000 the RFA during early negotiations. During 2000, we became aware for the first time of a safety issue regarding ABT-980. We discussed this previously unobserved safety issue internally for several weeks, and consulted independent experts about our concerns. Ultimately, as a result of these safety concerns, we decided to discontinue development of the compound. I understand that Hancock was notified of the termination and the parties agreed to replace it with other compounds.

ABT-518

15. In 2000 and 2001, I was generally responsible for the development of ABT-518. I was kept informed of the development of that compound by Dr. Perry Nisen, the Vice-President of Oncology Development, who reported directly to me, and Dr. Azmi Nabulsi, the Venture Head for the Oncology Venture, who reported to Dr. Nisen. In 2000 and 2001, I met and corresponded frequently with Dr. Nisen, Dr. Nabulsi, and other members of the ABT-518 development team to discuss the status and the ongoing clinical trial for the compound and I attended several executive-level meetings during which the status of ABT-518 was discussed. I also received the monthly status project reports created by the ABT-518 development team during this period.

16. ABT-518 is a oncological pharmaceutical compound that was under development at Abbott in 2000 and 2001. It belongs to a novel class of compounds known as Matrix Metalloproteinase Inhibitors (“MMPIs”). Matrix Metalloproteinases

("MMPs") are a family of enzymes that degrade a wide range of matrix protein substrates. High expression of these enzymes occurs in cancer and is associated with tumor growth. At the time of the agreement with Hancock, within Abbott's oncology venture, and the oncology field in general, it was hypothesized that inhibition of certain MMPs would inhibit tumor progression. Based on the preclinical work that Abbott had performed, we believed ABT-518 had certain potential advantages over other MMPIs. ABT-518 was highly selective for inihibition of two particular MMPs, gelatinase A and B, which were believed to play a particularly important role in tumor progression. Other MMPI compounds under development by Abbott's competitors — such as Pfizer's compound Prinomastat, and British Biotech's compound, Marimastat — were less selective in inhibition of these particular enzymes, although Prinomastat was moderately selective and most similar to ABT-518 in that regard. Therefore, we believed that ABT-518 might be more efficacious in inhibiting tumor progression. In addition, the competitors' MMPI compounds had exhibited side effects characterized by joint pain and stiffness ("joint effects" or "joint toxicity") in clinical trials, which limited the doses at which they could be administered. We hypothesized that these joint effects were caused by inhibition of MMPs other than gelatinase A and B, such as fibroblast collagenase. Because of ABT-518's greater selectivity, we believed that ABT-518 was less likely to cause joint toxicity, and that we might therefore be able to administer at higher doses that would be more efficacious. In addition, because of ABT-518's unique pharmacological properties, our preclinical work suggested that it could achieve more sustained and consistent potency, which might also allow greater efficacy without the need for dosing at levels that might cause greater toxicity.

17. As reflected in the first Annual Research Plan for ABT-518 that was provided to Hancock as part of the Agreement, from the beginning of the development of ABT-518 through 2000 we had spent $40 million on developing the compound. Attached hereto as D's Exhibit Y is a true and correct copy of the Research Funding Agreement dated March 13, 2001, which reflects Abbott's spending through 2000 at page JH008127.

18. The final Descriptive Memorandum for ABT-518, which I reviewed as discussed above and which was provided to Hancock as part of the Agreement, disclosed that "Abbott's Matrix Metalloproteinase Inhibitor (MMPI) program represents a novel therapeutic class, with the potential to alter the way that cancer is treated by preventing or modifying disease progression and/or metastases." D's Exhibit Y at JH008194. The Descriptive Memorandum also disclosed the problems experienced by competitor pharmaceutical companies' MMPIs including that (1) Marimastat had shown "no survival advantage [in pancreatic cancer]" and that other MMPI compounds had not demonstrated efficacy; (2) the competitor compounds, including Marimastat and Prinomastat, had "dose limiting toxicity" that "almost certainly preclude their long-term use, limit compliance and reduce optimal efficacy"; and (3) "Bayer recently dropped development" of its MMPI compound due to concerns about potential toxicity. *Id.* at JH008197-99. The Descriptive Memorandum also states that because ABT-518 was at a less advanced stage of development the "[side effect] hurdles will be even higher for this compound." *Id.* The Descriptive Memorandum also disclosed:

> As the 3rd or 4th MMPI to market, Abbott's compound will need to demonstrate a meaningful clinical advantage over compounds that are in more advanced development. Strict Go/No Go criteria will determine if the MMPI can

> meet these hurdles. If they cannot be met, the compound will not move forward.

Id. at JH008199. I believed at the time that I reviewed the final Descriptive Memorandum for ABT-518 and when it was provided to Hancock in March 2001 that it was accurate and contained the information necessary to accurately convey the condition of and prospects for the ABT-518 program.

19. Based on the information that was provided to me by members of the ABT-518 development team, I was optimistic in March 2001 about the prospects for development of ABT-518. I remained optimistic about ABT-518 into May 2001. During that month, at the American Society of Clinical Oncology ("ASCO") conference, we received newly-disclosed clinical trial data regarding certain MMPI compounds developed by other pharmaceutical companies that was pertinent to ABT-518. MMPIs were a novel pharmacology and no MMPI compound had successfully completed phase III trials or gained regulatory approval as of early to mid-2001. While we were optimistic about the MMPI program, it was an early-stage program in a novel class of compounds.

20. As discussed above, prior to May 2001, based on the limited information we had, I believed that Abbott had an opportunity to be successful where several of our competitors had not been successful because the pre-clinical work we had done to that point promised the possibility of developing a compound that had a selectivity profile that would inhibit the appropriate target MMP enzymes, gelatinese A and B, while not affecting other MMPs implicated in the toxicity observed in prior clinical tests. In other words, again based on the limited information available to us, I believed that ABT-518 was sufficiently different from its potential competitors that it had a good chance of avoiding the difficulties that we understood other pharmaceutical companies had

apparently encountered with their MMPIs. My view in this regard was based on information provided to me by the ABT-518 development team.

March 7-9, 2001 Portfolio Review Meeting

21. At the end of 2000, Abbott acquired the Knoll Pharmaceutical Division of BASF Corporation, together with compounds that Knoll was developing. On March 7-9, 2001, I attended a series of meetings regarding the pharmaceutical compounds in Abbott's portfolio and in the newly acquired Knoll portfolio as part of a portfolio review. The meetings were held at the Deerfield Hyatt Regency. Attached hereto as D's Exhibit 621 is a true and correct copy of the final schedule for the portfolio review that I received prior to the portfolio review. The purpose of the March 2001 Portfolio Review Meeting was to examine the technical, scientific, medical and commercial status of Abbott's pre-acquisition compounds and the compounds acquired through the Knoll acquisition.

22. During the Portfolio Review Meeting, Dr. Nisen gave a short presentation regarding ABT-518. I attended the presentation regarding ABT-518 and am familiar with the slides that Dr. Nisen included in that presentation. Attached hereto as D's Exhibit 782 is a true and correct copy of the slides presented during Dr. Nisen's ABT-518 presentation. As stated in the slides, the ABT-518 development team believed that there was "no joint-toxicity expected" with respect to ABT-518. In this respect we believed ABT-518 had a potential advantage over competitors' MMPI compounds, which had exhibited side effects of musculoskeletal pain and stiffness in the joints ("joint effects" or "joint toxicity"). As stated in the slides, we also believed that ABT-518 was "[h]ighly selective for the inhibition of gelatinases A & B" (which play a role in tumor progression), "very potent", and "potentially best in class." Although we believed at the

time these properties of ABT-518 would give it an advantage over other compounds, we did not have sufficient data to know that ABT-518 would succeed in clinical testing or differentiate from competitor compounds with any certainty. Thus, the slides presented by Dr. Nisen noted that "competitor data may pose additional development hurdles." *Id.* at ABBT0064326. The information Dr. Nisen presented regarding ABT-518 at the Portfolio Review Meeting accurately reflected the state of our knowledge about ABT-518 and about competitors' MMPI compounds at that time.

23. Shortly after Dr. Nisen gave his presentation on ABT-518 at the March 2001 Portfolio Review Meeting, I attended a much smaller, executive level meeting with Dr. Leiden and others. During that meeting, Dr. Leiden issued a directive to put a temporary hold on the Phase I M00-235 clinical trial of ABT-518, which was the "first in man" study of the compound. Dr. Leiden said that he wanted to wait until after the May 2001 ASCO conference before enrolling patients in this trial. At that conference, competitors were expected to release recent detailed clinical data regarding their MMPI compounds. Dr. Leiden stated that he wanted us to be able to analyze that data and its implications for the development of ABT-518 before continuing with the M00-235 trial and incurring additional expenses. At the time Dr. Leiden issued this temporary hold directive, enrollment had begun for the M00-235 trial.

24. Since Dr. Leiden had only recently joined Abbott, he was not familiar with the details of the ABT-518 program at the time of the March 2001 Portfolio Review Meeting. Those of us who were more familiar as of March 2001 with ABT-518 than Dr. Leiden, including myself and Dr. Nisen, disagreed with his decision to put a temporary hold on beginning the M00-235 clinical trial.

25. After the meeting discussed above, during which Dr. Leiden informed us that the clinical trial would be put on a temporary hold, I had a follow-up conversation with Dr. Leiden during which I explained to him why I believed the hold on the ABT-518 clinical trial should be lifted. I emphasized that the ABT-518 discovery and development teams advanced the drug based on pre-clinical data distinguishing it from competitor drugs. Furthermore, I explained our hypothesis that these pre-clinical differences might lead to meaningful clinical differences in clinical trials in patients. I suggested that we should not delay the ABT-518 clinical trial while waiting for release of data regarding other MMPI compounds because even if that data was negative, it might not bear directly on ABT-518. I pointed out that a delay in the clinical trial could later put Abbott at a competitive disadvantage if we continued development after the ASCO conference. I also informed Dr. Leiden that the amount of money we would save on the clinical trial was relatively minor compared to the value to Abbott that would be lost if the launch of the compound was delayed. I also recall reminding Dr. Leiden that Hancock was a partner for ABT-518 because I thought it might alleviate some of his concerns regarding the financial risks of continuing with the development of the compound without waiting for the ASCO results.

26. A short time after Dr. Leiden had placed the hold on the clinical trial for ABT-518, he informed us that he had reversed his decision and that he had lifted the temporary hold on the clinical trial. Given the short time that the hold was in place and the fact that the clinical trial did, in fact, continue after the hold was lifted, I did not believe at the time, nor do I believe now, that the temporary hold had a material impact on Abbott's development of ABT-518 or on the prospects for the compound's success.

27. I attended a Prioritization Review during the first week of May 2001 that was chaired by Dr. Leiden. The May Portfolio Prioritization was designed to examine the medical needs in particular therapeutic areas and to determine what opportunities may exist for Abbott in those areas. I attended the presentation given by the Oncology Venture during the early May Prioritization Review. Since the data from the ASCO conference was not yet available, we did not make a decision regarding ABT-518 during that Prioritization Review. Attached hereto as D's Exhibit 755 is a true and correct copy of the Oncology Venture presentation given during the May 2001 Portfolio Review. As reflected in the document, the presentation by the Oncology Venture was a general overview of the venture's work and did not include any new information about ABT-518, since the May 12-15, 2001 ASCO conference had not yet taken place. I have no recollection that there was any discussion at the early May retreat about the temporary hold that Dr. Leiden had placed on the M00-235 trial in March or of his decision to lift that temporary hold. Nor do I remember any discussion about the possibility of terminating ABT-518.

ASCO Results and Discontinuation of ABT-518

28. Prior to the ASCO conference, we had only very limited information from publicly available sources, such as press releases, regarding some of the recent competitors' trials of MMPI compounds. For example, attached hereto as Exhibit FL is a true and correct copy of an email produced from Abbott's files with an August 7, 2000 press release from Pfizer announcing that it was halting Phase III trials of Prinomastat in combination with standard chemotherapy in patients with non-small cell lung cancer and advanced hormone refractory prostate cancer because they "did not meet primary

efficacy objectives" and that "neither detrimental nor convincing beneficial effect was observed." The press release noted that Pfizer "intends to continue exploration of Prinomastat in other tumor types and, most importantly, in earlier stage disease, where oncologists believe inhibition of angiogenesis may have greater utility." The release also noted that "four phase II trials are currently underway and two additional phase II trials will begin shortly." We could not make any determination from press releases and other publicly available reports, however, regarding the potential impact of this information regarding competitor's trials on ABT-518. For example, the press reports did not identify what "primary efficacy objectives" were being measured in the clinical trials of Prinomastat or disclose whether the trials also measured achievement of secondary efficiacy objectives, which could be important in analyzing whether there were signals of efficacy in more sensitive efficacy end points. Nor did the press reports provide other essential details of the studies, such as sample size, disease stage and other patient characteristics. In order to make a determination regarding the potential significance of the competitors' trials with respect to ABT-518, it was essential for us to review and analyze the peer-reviewed reports of the actual clinical data, which was released at ASCO.

29. ASCO is the leading clinical oncologic group in the United States and its yearly conference is probably the most important oncology conference in the world. Abbott employees attend the ASCO conference each year, in part to learn new information about developments in the field, including peer-reviewed reports of new clinical data regarding oncology compounds in development by other companies. I understand that ASCO rules provide that presentations at the conference cannot include

previously released data, therefore, by necessity, all the data released at ASCO each year is new.

30. Dr. Nisen, Dr. Nabulsi, Dr. Davidsen, and other Abbott employees attended the May 12-15, 2001 ASCO conference. As discussed above, since we were in the midst of the ABT-518 development program in 2001, we were particularly interested in the information about MMPIs that would be presented during the conference. We were aware that other pharmaceutical companies would be presenting significant amounts of previously unavailable clinical trial data at that conference regarding their respective MMPI programs. We expected that multiple competitors to reveal their detailed results regarding studies of various MMPI compounds with differing properties and toxicity profiles tested on multiple tumor types under a variety of different circumstances, including in combination with other treatments and as stand-alone (i.e., "mono") therapy and in advanced Phase III trials. As we had discussed in meetings at Abbott prior to the conference, we believed the information that would be disclosed during the ASCO conference would be instrumental in allowing us to make an informed decision regarding the development of ABT-518.

31. On May 22, 2001, I received from Dr. Nisen a summary of the findings on competitors' MMPIs that had been presented at the May 2001 ASCO conference and the ABT-518 project team's recommendations regarding ABT-518, based on this new information. Attached hereto as D's Exhibit 586 is a true and correct copy of the 2001 ASCO MMPI Update that I received from Dr. Nisen. On or around May 28, 2001, I attended a presentation at Abbott made by Dr. Nisen that summarized the MMPI competitor clinical trial data that was released during the conference. Dr. Nisen also

provided additional details regarding the clinical trial in an oral presentation and question and answer session that accompanied his report.

32. Attached hereto as D's Exhibits FI, 793, and FK are true and correct copies of documents I recognize to be abstracts and posters from the 2001 ASCO conference, which were summarized in Dr. Nisen's report. As reflected in the abstracts, more detailed data was released at ASCO than had previously been available. For example, as reflected in D's Exhibit 793 at ABBT0556352, Pfizer's abstract for its Phase III prostate cancer trial for Prinomastat reports the large size of the study ("553 [patients] were enrolled; interim results were available for 406 [patients]"), the characteristics of the patients with respect to age and disease severity ("balanced with median age 71 years, median PSA 94 ng/mL and 33% measurable disease"). ("PSA" stands for prostate specific antigen, which is a protein measured to track disease progression.) The abstract also reports that in the Prinomastat prostate cancer trial "Grade-2 MS [musculoskeletal effects] were observed in 13, 22, and 22% of the [patients] in the placebo, 5 and 10 mg arms, respectively." While Pfizer had previously reported merely that "primary efficacy objectives were not met" in its study of Prinomastat in prostate study cancer patients, in the ASCO abstract it reported the results of the specific primary and secondary endpoints ("[n]o differences were observed among the treatment arms in PSA response rate (RR, 75% reduction for 3wks), progression-free survival by radiography (RPFS), PSA (50% increase for 3wks), or symptoms (SPFS); or overall (OS) and 1-year survival").

33. With respect to Pfizer's Phase III trial of Prinomastat in patients with non-small cell lung cancer study, as reflected in D's Exhibit 793, the abstract reported the large size of the study ("686 [patients] were enrolled; interim results are available for 677

[patients]"), the characteristics of the patients with respect to age, sex, disease severity, and type of tumor ("balanced with median age 62 years, 62% male, 85% WHO PS 0/1 [a measurement of disease severity], 56% adenocarcinoma [a type of tumor], 12.6% stage IIIB(T4), 74% stage IV, 11.8% recurrent disease, and 84% measurable disease"). *Id.* at ABBT0556350. It reported the joint toxicity experienced by treatment group ("Grade-2 [musculoskeletal] events occurred in 16, 19, 22, and 31% of [patients] in placebo, 5, 10, and 15 mg arms, respectively"). Finally, while Pfizer's press release had merely reported that "primary efficacy objectives were not met", the abstract reported the results with respect to specific primary and secondary endpoints ("[n]o differences were observed among the treatment arms in overall (OS) or 1-year survival, progression-free survival (PFS), symptomatic PFS (SPFS) or response rate"). *Id.*

34. As reflected in D's Exhibits FK and FI, Pfizer also published at ASCO the clinical data from a smaller, earlier phase trial of 44 patients with metastatic breast cancer. Even though this trial included a higher 25 mg BID dose, as well as a 5 mg dose, Pfizer reported that "[n]o objective disease responses were observed" and that "[m]edian [time-to-progression] was 8 weeks in both arms". D's Exhibit FK at ABBT0556332; D's Exhibit FI at ABBT0556331.

35. Bayer and British Biotech also reported negative results at ASCO in trials of their compounds, BAY 12-9566 and Marimastat. For example, as reflected in D's Exhibit 793, Bayer released clinical data from a study of 243 patients, which showed "no evidence of an impact of BAY [12-9566] on [progression-free survival or [overall survival]." *Id.* at ABBTABBT0556354.

36. After we reviewed the new information about MMPIs that was disclosed at ASCO, we concluded that Abbott should not continue with the development of ABT-518. The clinical trial data released at ASCO, involving a variety of compounds, tumor types, patient characteristics, disease severity, combination therapy and mono therapy, failed to show significant signals of efficacy. The clinical data released at ASCO regarding Prinomastat was particularly significant, because Prinomastat overlapped with ABT-518 in terms of its selectivity for gelatinase A and B, and was tested in large scale advanced clinical trials. The key finding, based upon the ASCO data, was that MMPIs were less likely to demonstrate efficacy than we had hypothesized. In addition, in light of the additional data regarding joint toxicity experienced by competitors, there remained uncertainty regarding whether ABT-518, despite its different characteristics, would be able to avoid these problems. I concluded based on the clinical data that was disclosed at ASCO, particularly the lack of significant signals of efficacy, that it was much less likely that we would be able to successfully develop ABT-518. Some members of ABT-518's oncology team, including Dr. Nisen, advocated continuing clinical trials to test whether ABT-518, despite the negative data released at ASCO, might still be able to distinguish itself from the other competitors. I believed, however, that in light of the overwhelming negative data released at ASCO, the chances of success were too low to justify continuation of funding for development of ABT-518 at that time. I therefore concurred in the decision made by the Pharmaceutical Executive Committee ("PEC") shortly after the May 28, 2001 presentation by Dr. Nisen, that Abbott should not proceed with the compound.

37. Even though we had decided to terminate the ABT-518 program, we had an ethical obligation to the patients already enrolled in the M00-235 clinical trial. Therefore, we decided to allow the patients that were already enrolled in the clinical trial to complete the trial.

Out-Licensing of ABT-518

38. After we decided to terminate the development program of ABT-518 in late May or early June 2001, I encouraged Abbott's business development team to look for potential out-licensees or support for co-development of the compound. While I was not personally involved in the effort to out-license the compound, I was aware that it was ongoing. For example, I was informed that we made a presentation to Goodwin Philanthropy in an attempt to interest that organization in funding additional clinical trials for ABT-518. Attached hereto as D's Exhibit DT is a true and correct copy of the email exchange between Dr. Leiden and Dr. Nisen that was forwarded to me regarding the draft presentation for Goodwin Philanthropy.

39. I was generally aware of the out-licensing efforts of the business development team for ABT-518. I learned that the team had contacted and provided information about the compound to several pharmaceutical companies, including Chiron, Paramount Capital, Salmedix, and Sunessis, as well as Duke University. I was informed that the companies had no interest in licensing ABT-518 from Abbott.

ABT-594

40. In 2000 and 2001, I was generally responsible for the development of ABT-594. I was kept informed of the development of that compound by Dr. Chris Silber and Dr. Bruce McCarthy during that time period. Dr. Silber was the Venture Head of the

Analgesia Venture until February or March 2001 when Dr. McCarthy was promoted to Venture Head. During the earlier time period, Dr. McCarthy was the medical director of the ABT-594 development team. In 2000 and 2001, I often met and corresponded with Dr. Silber, Dr. McCarthy, and other members of the ABT-594 development team to discuss the status and the ongoing clinical trials for the compound. I also attended several executive-level meetings during which the status of ABT-594 was discussed. I also received the monthly status project reports created by the development team during this time period.

41. ABT-594 is a pharmaceutical compound that was under development in the Analgesia Venture at Abbott from 1997 through October 2001. As reflected in the first Annual Research Plan that was provided to Hancock, through 2000, Abbott had spent $97.3 million developing ABT-594. D's Exhibit Y at JH008121.

42. ABT-594 falls within the class of pharmaceutical compounds known as cholergenic channel modulators ("CCM") or neuronic nicotinic receptors ("NNR"). Nicotine, the active agent in cigarettes, has been shown to have activity in psychosis, analgesia, cognition, depression, and a variety of other potential disease states. In an attempt to build on the observations that had been made over the years about the pharmacology associated with nicotine, Abbott had a long-standing NNR project that attempted to get the desired effects of nicotine without using nicotine itself and to develop compounds that did not exhibit the typical side effects of nicotine, namely, dizziness and nausea.

43. As discussed above, I reviewed the November 2000 Descriptive Memoranda that were provided to Hancock. I cannot recall whether I reviewed the final

February 2001 Descriptive Memorandum for ABT-594 before the RFA was executed. The final February 2001 Descriptive Memorandum for ABT-594 that was provided to Hancock disclosed that the likelihood of ABT-594 reaching its target profile of low nausea/vomiting was "Low". D's Exhibit Y at JH008172. It also disclosed that during previous clinical trials, the "most common adverse events for subjects receiving 75 ug [micrograms] BID [twice-a-day] were nausea (15%), headache (13%), dizziness (7%), insomnia (6%), and vomiting (5%)." *Id.* at JH008171. The Descriptive Memorandum further disclosed that the therapeutic window (i.e., the ratio between the maximum tolerated dose and the minimum efficacious dose) might be small because the Phase IIa studies "suggest a trend towards analgesic effect [efficacy]" at 75 micrograms twice-a-day and the Phase I studies indicated that the maximum tolerated dose might be as low as 150 micrograms per day). *Id.* at JH 008171. The Descriptive Memorandum also disclosed that a Go/No Go decision for clinical efficacy was expected in June 2001 at the conclusion of the Phase IIb (dose-ranging) trial ("M99-114"). *Id.* at JH008166.

M99-114 Trial

44. By 2000 the ABT-594 development team had completed a number of Phase I and small Phase II clinical trials. In April 2000, we initiated the Phase IIb M99-114 clinical trial for the treatment of painful diabetic neuropathic pain. The M99-114 trial was a dose-ranging study designed to determine the therapeutic index for ABT-594. The therapeutic index for a pharmaceutical compound is the relationship between the lowest dose that demonstrates efficacy and the highest dose that has an acceptable side effect profile. While there were other clinical trials planned for ABT-594, the M99-114 dose-ranging study was a necessary precursor to the additional clinical trials that were to

be conducted for the compound. The M99-114 trial was designed with four different arms or dose groups: placebo, 150 mcg BID, 225 mcg BID, and 300 mcg BID. The primary purpose of the study was to determine a dose-response curve; this entails selecting a range of ascending doses to eastablish the safety and efficacy associated with each dose. High doses are selected wth the intention of identifying a maximal dose either by demonstrating no further gain in efficacy or finding unacceptable adverse events irrespective of the efficacy. We expected that at the higher doses there would be more drop-outs from side effects than at lower doses because the higher doses had been selected specifically to define an adverse event profile for the compound.

45. The M99-114 clinical trial was double-blinded and placebo controlled. A double-blinded, placebo-controlled clinical trial is one in which neither patients nor physicans know which dose of drug, if any, is being administered to each patient. The sponsor of a double-blinded, placebo-controlled trial, such as Abbott in the case of the M99-114 study, is also unaware of what dose is being administered to each patient.

46. Based on the earlier clinical trials conducted for ABT-594, we knew that the main dose-limiting side effects of ABT-594 were nausea, vomiting and dizziness. However, it was unclear whether there was a dose of ABT-594 that would be both efficacious and sufficiently well-tolerated to be a viable drug. The purpose of M99-114 was to determine if a dose with adequate efficacy and an acceptable side effect profile exisited. During the M99-114 clinical trial, we learned before the data was unblinded that there were a number of patients who dropped out of the trial due to side effects. The fact that there were drop-outs as a result of side-effects was not unexpected, since the trial had been designed to determine the dose-limiting side effects. Moreover, we could not

know before the data was unblinded whether the drop-out rate indicated that the therapeutic window for the drug would be too narrow for the drug to be commercially viable because, before unblinding, we did not, and could not know at what doses the drop-outs were occurring. We believed at the time, for example, that it was possible that the adverse side effects were occurring at the highest doses and the lowest dose of 150 mcg would likely be both efficacious and well tolerated.

47. In late 2000, I became aware that we were experiencing slower enrollment in the M99-114 trial than we had expected and planned. As a result of this enrollment rate, the ABT-594 project team assessed whether we could still achieve our clinical goals for the M99-114 trial if we enrolled fewer than the originally targeted number of patients. I was kept informed of the analysis and was personally involved in reviewing the team's assessment. For example, attached hereto as D's Exhibit DB is a true and correct copy of a notice of a December 11, 2000 meeting that I attended with Chris Silber and David Morris, one of the Abbott statisticians working on the M99-114 trial, in order to discuss the power calculations for the M99-114 clinical trial. Based on all the information provided to me by the ABT-594 team, I made the decision to stop enrollment in the M99-114 trial before the trial achieved the original target number of patients. I made this decision because I had been informed and I had concluded that stopping enrollment at a smaller number than originally planned would not negatively affect in a meaningful way our ability to have a successful trial that would provide us with sufficient information to make an informed decision on the future of the product. I was informed by the clinical statisticians, for example, that they believed that the trial would likely reach a statistically significant endpoint with the number of patients that we had already enrolled in the trial.

Based on this information provided to me by the ABT-594 team, and my experience with clinical trials, I concluded that we would gain little incremental information from reaching the target enrollment number than we would gain from the enrollment we had already achieved, and that the further delay and expense that would be caused by attempting to reach the original enrollment goal was unwarranted. My decision in this regard was informed by the fact that, while we always try to enroll the target number of patients in a clinical trial, it is not uncommon, especially in the case of larger clinical trials such as M99-114, that we do not reach that target number and yet we are still able to achieve a statistically significant result that we are able to use in making decisions about the future of our products.

48. I was aware in late 2000 of efforts to explore a potential partnership with other pharmaceutical companies to co-develop ABT-594. The decision to seek a potential partnership was to gain additional resources to help fund the development of ABT-594 and maximize the value of the compound to Abbott and was not based on any belief that the compound would be discontinued. We would not have sought additional funding to co-develop ABT-594 and continued to spend millions of dollars of Abbott's research and development budget on the development of the compound if we believed that we were going to discontinue the development of the compound.

49. I am aware that Hancock has claimed that the fact that the clinical study report for the M99-114 clinical trial contains a statement that there were "significant changes in the developmental strategy" for the compound indicates that Abbott had decided to discontinue the development of the compound even before the M99-114 trial was completed. Hancock's contention is incorrect. No significant changes were made to

the developmental strategy for ABT-594 until after the results of the M99-114 clinical trial was unblinded and the results of that trial were analyzed by the ABT-594 development team and senior management in the summer and early Fall of 2001.

50. In early 2001, at the end of the enrollment of the M99-114 clinical trial, we were still very optimistic about the prospects of ABT-594 and considered ABT-594 to be among our top ten development prospects in January 2001. For example, the company's January 2001 portfolio analysis review material notes that we considered ABT-594 one of our top four projects -- the same place it held in our July 2000 analysis. Attached hereto as D's Exhibit 750 is a true and correct copy of the January 2001 Review Reference Materials. *Id.* at ABBT0012382.

51. On February 2, 2001, I attended an ABT-594 Project Review presentation that was given to update the PEC on the status of the ABT-594 development program. Attached hereto as D's Exhibit 748 is a true and correct copy of the slides from that presentation. As reflected in the presentation, as of February 2, 2001, we believed that ABT-594 would be "First-in-class". *Id.* at ABBT0002361. The presentation also reflects that enrollment in the M99-114 clinical trial ended on January 5, 2001 with 269 patients and that the width of confidence intervals was not meaningfully different going from 269 to 320 patients (the target enrollment number). *Id.* at ABBT0002433. As we had determined in the fall of 2000, the difference between 269 and 320 patients was not significant in terms of our confidence in achieving statistically significant results for the trial. I do not remember any discussion during that presentation of blinded data from the trial or of the drop-out rate of the trial. Nor do I remember anyone who attended the

presentation expressing concern that ABT-594 would not be a successful compound based on that clinical trial.

52. Prior to the unblinding of the data from the M99-114 trial, we did not know whether there was a dose at which the compound would be both efficacious and have an acceptable side effect profile. We did not know prior to the unblinding of the M99-114 trial whether the drop-out rate for the M99-114 trial meant that the product would not be successful or that ABT-594 would be a "probable teriminate". I had not made any such determination personally and no one at Abbott ever informed me that they or anyone else had done so.

March 7-9, 2001 Portfolio Review Meeting

53. I attended the ABT-594 presentation by Dr. McCarthy during the off-site Portfolio Review Meeting on March 7-9, 2001. Attached hereto as D's Exhibit 620 is a true and correct copy of the slides for ABT-594 presented during the portfolio review. I do not recall any discussion during the ABT-594 presentation regarding the drop-out rates for the ongoing M99-114 trial and at no point during that presentation did we come to a consensus that ABT-594 would probably be terminated. Since the results of the M99-114 trial had not yet been unblinded in March 2001, it was unclear what the development prospects for the compound were going to be until after April 2001 when the results would be unblinded and analyzed.

The Results of the M99-114 Clinical Trial and the Discontinuation of ABT-594

54. The blind on the M99-114 trial was broken on April 23, 2001. Attached hereto as D's Exhibit DN is a true and correct copy of the Monthly Highlights for April 2001 that I prepared and circulated regarding the breaking of the blind on that date. As

reflected in the Monthly Highlights, the results of the trial were not available until the week of April 30, 2001. After the results of the M99-114 trial became available, the ABT-594 development team, under my supervision, began an intensive analysis of those results that lasted several months. We had not decided as of September 2001 whether we would continue the development of ABT-594 with a lower dose than the lowest dose that had been used during the M99-114 trial.

55. On October 5, 2001, I sent an email to several development team heads, including Dr. McCarthy, with a schedule for an upcoming PEC meeting. The PEC was created by Dr. Leiden sometime in 2001 and the committee reviewed key programs and key decisions on a monthly basis. I was in 2001 and currently am a member of the PEC. Attached hereto as D's Exhibit DF is a true and correct copy of the October 5, 2001 email I sent to Dr. McCarthy and others. As reflected in the email, ABT-594 was to be part of the discussion for the PEC meeting to be held on October 8, 2001. *Id.* at ABBT224538. On October 5, I received from Dr. Leiden an email stating that he wanted to include in the PEC meeting agenda a discussion regarding a possible additional dosing study for ABT-594. Attached hereto as D's Exhibit DG is a true and correct copy of the email I received from Dr. Leiden on October 5, 2001. During the October 8, 2001 meeting, the ABT-594 development team made a proposal for an additional dose-ranging clinical trial. After careful consideration, the PEC decided not to start a new dose-ranging trial and instead to out-license ABT-594. We concluded that although the M99-114 trial had established that ABT-594 was efficacious, the side effect profile that came with the compound would make it unattractive in the marketplace. This decision by the PEC

came after months of analysis of the M99-114 results and after we had considered numerous possibilities for pursuing alternative development pathways for the compound.

2001 Funding for ABT-594

56. The first Annual Research Plan for ABT-594, attached to the Agreement and provided to Hancock, disclosed that the "2001 Current Projection (Plan)" for ABT-594 spending was "35.0" million dollars, including $5.2 million for a Phase IIb Osteoarthritis study and $6.3 million for Phase III studies scheduled to start in 2001." D's Exhibit Y at JH008121-22.

57. In late 2000, I received a copy of the Analgesia Venture 2001 Plan from the Analgesia Venture. Attached hereto as D's Exhibit 759 is a true and correct copy of the Analgesia Venture 2001 Plan. In early 2001, I also received the Plan Final Reference Package that reflects Abbott's planned spending in 2001 for its various compounds. Attached hereto as D's Exhibit 616 is a true and correct copy of the March 2, 2001 Plan Final Reference Package. As reflected in both of these documents, Abbott had budgeted to spend $9.31 million on ABT-594 through the June 2001 Go/No Go decision. D's Exhibit 759 at ABBT144633.UR; D's Exhibit 616 at ABBT0037544.

58. Since the M99-114 clinical trial was a critical dose-ranging trial for ABT-594, we decided to wait for the results of the trial before beginning any other clinical trials for the compound. We had scheduled a Go/No-Go decision for ABT-594 for June 2001. A Go/No Go decision is a point in the development of a compound at which determinations are made as to whether to continue or terminate the development of the compound, or to otherwise change course with the compound. The Go/No Go decision for ABT-594 in June 2001 was to be based on the data from the M99-114 clinical trial

after it was unblinded in April 2001. Since the M99-114 clinical trial results would determine whether Abbott would continue to develop ABT-594, we decided to budget the ABT-594 program based on "milestone funding" through the clinical trial and "blue plan" funding of the work on the compound was planned subsequent to a Go Decision in June 2001. In Abbott's parlance, "blue plan funding" is funding that we expect to spend on a particular program depending on the outcome of a particular event. As reflected in the March 7-9, 2001 Portfolio Review Meeting presentation on ABT-594, we intended to spend an additional $5.6 million on ABT-594 in 2001 after the June Go/No Go decision. D's Exhibit 620 at ABBT0048644. Provided that the decision was made to continue the development of the compound, this additional money would be spent on a Phase IIb Molar Extraction clinical study, as well as on the prepratory work necessary to initiate Phase III and additional Phase I studies at the beginning of 2002. Attached hereto as D's Exhibit 749 is a true and correct copy of a December 21, 2000 Plan Assumption Memo reflecting additional funding for ABT-594 after June 2001.

59. The change in budgeted spending on ABT-594 in 2001 that resulted from our decision to milestone fund the program until the Go/No Go decision in June 2001 was more than off-set in the budget by greater expected spending in later years and was due to the fact that the only critical path activity for the compound for much of 2001 was the M99-114 clinical trial. During the March 2001 Portfolio Review Meeting discussed above, Dr. McCarthy presented the ABT-594 development team's expected spending on the compound in 2001 and later years. As reflected in the presentation, the development team expected to spend $59.6 in 2002 on developing the compound. D's Exhibit 620 at ABBT0048644. This amount is $14.6 million greater than the amount disclosed to

Hancock in the first Annual Research Plan for ABT-594 that was an exhibit to the Agreement. D's Exhibit Y at JH008121. The presentation also reflects that the development team expected to spend $55.7 million in 2003, a figure that was $23.7 million greater than the $32 million disclosed in the Agreement. D's Exhibit Y at JH008121; D's Exhibit 620 at ABBT0048644. The projected spending for 2004 was $21.8, $6.8 million greater than stated in the Agreement. D's Exhibit Y at JH008121; D's Exhibit 620 at ABBT0048644. For calendar years 2001 through 2005, we estimated spending $163.6 million on ABT-594, an amount $24.6 million more than the $139 million that was disclosed in the Agreement. D's Exhibit Y at JH008121; D's Exhibit 620 at ABBT0048644.

60. The change in budgeted spending for ABT-594 in 2001 would not have delayed the development of the compound had a "Go" decision been reached in June 2001, after the unblinding of the M99-114 trial data. To the contrary, as reflected in the March 2001 Reference Package discussed above, we expected to file a New Drug Application ("NDA") in September 2003, the same date that we had disclosed to Hancock in the first Annual Research Plan for ABT-594. D's Exhibit Y at JH008121; D's Exhibit 616 at ABBT0037544. The NDA is filed with the FDA to get approval for the commercialization of a pharmaceutical compound. The Reference Package thus confirms that we did not expect the reduction in planned ABT-594 spending for 2001 to cause any delays in the schedule for the development of the compound that we had disclosed to Hancock at the time the Agreement was executed. The Reference Package also reflects that the ABT-594 Phase III trials were to be delayed only from October 2001

to April 2002 (assuming a "Go" decision after the Phase IIb trial), and that this minor delay was not expected to affect the launch date for the compound.

Out-Licensing of ABT-594

61. Because of the narrow therapeutic index of ABT-594 that had been shown by the unblinded Phase IIb results, the prospects for outlicensing or selling ABT-594 were very low. For example, after the Phase IIb trial, there was no interest by Purdue Pharma, a company that had expressed interest earlier that year, in in-licensing the compound. I am aware that one potential licensee, Bayer Animal Health, expressed an interest in a potential license for ABT-594. It would have been commercially detrimental to out-license ABT-594 as a drug for animals, while Abbott was developing a compound with a similar mechanism for use in humans. Drugs that are on path to development for human use are usually not developed for animal use because of the significant commercial impact on the compound. I believe doctors generally are reluctant to prescribe their patients a drug that is being marketed for animals.

ABT-773

62. From 2000 through 2002, I was generally responsible for the development of ABT-773. Until April 2001, Dr. Carl Craft was the Head of the Anti-Infective Venture, which was responsible for the development of ABT-773. In or about April 2001, Dr. Stanley Bukofzer took over as Head of the Anti-Infective Venture. Both Dr. Craft and Dr. Bukofzer reported to Dr. Eugene Sun, Abbott's Division Vice-President, Global Pharmaceutical Research & Development, who in turn reported directly to me. I was kept apprised of the development of ABT-773 by Dr. Sun, as well as by Drs. Craft and Bukofzer and other members of the ABT-773 development team. From 2000

through 2002, I met and corresponded with Drs. Sun, Craft and Bukofzer, and with other members of the ABT-773 development team, to discuss the status and the ongoing clinical trials for the compound and I attended several executive-level meetings during which the status of ABT-773 was discussed. I also received the monthly status project reports created by the development team during this time period.

63. ABT-773 is a ketolide antibiotic compound that was under development by Abbott from 1997 through the summer of 2002. ABT-773 was being developed for four indications: acute bacterial exacerbation of chronic bronchitis, pharyngitis, community-acquired pneumonia, and acute bacterial or maxillary sinusitis. As reflected in the Agreement, through 2000, we had spent approximately $188.4 million developing ABT-773. D's Exhibit Y at JH008117.

64. At the beginning of 2001, ABT-773 was one of the top four projects under development at Abbott as noted in the 2001 Reference Package. D's Exhibit 750 at ABBT0012382. It was considered the fourth most valuable compound under development by our company based on its expected value. *Id.* at ABBT0012381.

65. As stated above, I reviewed the November 2000 Descriptive Memorandum for ABT-773 that was provided to Hancock. I cannot recall whether I reviewed the final February 2001 Descriptive Memorandum for ABT-773 before the RFA was executed. The final ABT-773 Descriptive Memorandum that was provided to Hancock as part of the Agreement disclosed that during a Phase II trial conducted in 1999, 1% of patients taking both the 100 mg and 200 mg TID (three times a day) doses of ABT-773 experienced elevated liver function tests. D's Exhibit Y at JH008156. With regard to the dosing of ABT-773, the first Annual Research Plan for ABT-773 that was

provided to Hancock as part of the Agreement disclosed that tablet dosing for ABT-773 would be "150 mg QD [once-a-day] or 150 mg BID [twice-a-day] dosing based on severity of indications." D's Exhibit Y at JH008117. With regard to the ABT-773 pediatric program, the Annual Research Plan for the compound states that the indications for ABT-773 are "Adult Tablet" and "I.V." and that disclosed that we did not plan to spend any money on pediatric or taste testing studies for the oral formulation in 2001. D's Exhibit Y at JH008117-18. The ABT-773 Descriptive Memorandum states that an "oral formulation" would "enabl[e] penetration" into the pediatric market but makes no representations regarding the timing of the program. D's Exhibit Y at JH008153-58.

66. ABT-492, a quinolone anti-infective, was another compound included in the Hancock Agreement's basket of compounds. D's Exhibit Y at JH008179-85. The final Descriptive Memorandum for ABT-492 that was provided to Hancock as part of the Agreement, disclosed the potential for both QT prolongation and liver toxicity for the entire quinolone class of antimicrobials:

> The quinolone class has potential prolongation of the QT interval and other cardiovascular effects. There is also increased regulatory scrutiny due to recent quinolone withdrawals from international markets. ABT-492 has been evaluated in the standard in vivo models used to evaluate QT interval potentials of other antibiotics and has shown no evidence of increasing QT. Also, compared to marketed quinolones, preclinical studies show no evidence or no increased incidence of . . . liver toxicity.

Id. at JH008184. I was not informed by anyone that Hancock was concerned about this disclosure or that it made ABT-492 less desirable to Hancock during the negotiation and finalization of the Agreement.

<u>There Were No QT Prolongation Issues with ABT-773 as of March 2001</u>

67. I was aware in 2000 and 2001 that it was well known that, in some circumstances, high doses of macrolide anti-infectives could have QT prolongation

effects. Even though macrolides, and other types of anti-infectives, including quinolone antibiotics, are known to have the potential for such issues, anti-infectives are widely used and prescribed to patients. I was also aware during this period that the FDA was concerned generally with the potential for QT prolongation in all drugs in development, including anti-infectives. Since ketolides are a class of compounds related to macrolides, I was further aware that the FDA was paying attention to ketolides with regard to these issues and that Abbott, like other drug sponsors, would have to present data to the FDA about ABT-773 sufficient to satisfy the Agency that the compound was safe with respect to QT prolongation.

68. I attended the End of Phase II meeting with the FDA on November 27, 2000. Attached hereto as D's Exhibit 762 is a true and correct copy of the Memorandum of Meeting Minutes that reflects my attendance at that meeting. Attached hereto as D's Exhibit 582 as a true and correct copy is also the FDA contact sheet from that meeting that I received after the meeting. My recollection is that during that meeting there was general discussion about how to demonstrate the absence of a meaningful QT prolongation signal by the FDA and that the FDA indicated that Abbott would need to show that there was no QT prolongation problem with the compound. However, the FDA did not indicate that it had seen any evidence of a QT prolongation issue with the clinical data of ABT-773. At this time, the FDA was creating guidelines for assessing potential QT prolongation effects of all drugs that were being investigated for approval.

69. On or about December 5, 2000, I attended a project review presentation for ABT-773 with Dr. Leiden. The presentation was designed to provide an overview of the ABT-773 project for Dr. Leiden. Attached hereto as D's Exhibit 787 is a true and

correct copy of the December 2000 presentation for ABT-773 that I attended. As noted in the presentation, and as discussed above, we had not observed a consistent QT effect at the clinical doses of ABT-773; the effect noted in the presentation was observed during Phase I studies for doses greater than 800 mg, a dose far higher than any that would be prescribed to patients. *Id.* at ABBT205202. Based on these results, we did not believe, as of December 2000, that ABT-773 had a QT prolongation issue. The plan going forward was to monitor QT prolongation in the Phase III along with all other routine clinical assessment studies that were being initiated.

70. On February 12, 2001, I attended a presentation to the PEC by Dr. Craft designed to update the PEC on the ABT-773 program. Attached hereto as D's Exhibit 607 are true and correct copies of the slides presented to the PEC by Dr. Craft on February 12, 2001. As reflected in the presentation, as of February 12, 2001, we had not observed a QT prolongation issue with ABT-773 at the doses at which the drug would be prescribed to patients. D's Exhibit 607 at ABBT0576844. We recognized that QT prolongation was an issue that the FDA was interested in with all classes of drugs. We therefore wanted to ensure that we met the FDA's expectations with regard to the quantity and quality of the data we collected during our clinical trials. However, since we had not seen data reflecting a QT prolongation issue with ABT-773, we did not believe it would be any more difficult for the ABT-773 program to satisfy the FDA with regard to QT prolongation that it would be for any other drug development program.

71. I attended a presentation regarding ABT-773 that Dr. Craft gave at the Portfolio Review Meeting that took place from March 7-9, 2001. Attached hereto as D's Exhibit 622 are true and correct copies of the slides presented by Dr. Craft. Dr. Craft did

not present any information at that meeting that led me to believe that there was a QT prolongation issue with ABT-773. *Id.* at ABBT0013212-13. As discussed above, we were aware of the FDA's concern regarding the potential for QT prolongation issues in macrolides and ketolides as a class, but there was no evidence available to us that the compound would have clinically significant issues with QT prolongation.

72. On March 19, 2001, I attended a follow-up presentation to the PEC regarding ABT-773. Attached hereto as D's Exhibit 631 is a true and correct copy of the slides for that presentation. As noted in the presentation, there was no new information regarding the potential for QT prolongation issues in ABT-773 of which we were aware by that date. *Id.* at ABBT120480.UR.

73. In sum, during early 2001, in the period before the Hancock Agreement was executed, I was not aware of any significant unresolved issues for ABT-773 with regard to QT prolongation. While I was aware that there was a general concern at the FDA with regard to QT prolongation issues for all new drugs as well as for all anti-infectives, none of the clinical data that had been observed with regard to ABT-773 at that point raised a concern because we had not observed a QT prolongation issue at the doses we expected to prescribe to patients.

<u>There Were No Liver Toxicity or Hepatotoxicity Issues with ABT-773 as of March 2001</u>

74. As with QT prolongation issues, I was aware in 2000 and 2001 that the FDA was generally concerned with liver toxicity -- also known as hepatotoxicity -- with regard to many different types of drugs With regard to ABT-773, I was also aware that in an earlier clinical trial we had observed elevated liver enzymes, biochemical markers for liver toxicity, in a few Japanese subjects who had participated in a single small Phase I

study conducted as part of the Japanese ABT-773 development program. The Japanese subjects in this trial were residents of Hawaii. I was informed by the ABT-773 team that the results of this study with regard to liver toxicity were not consistent with our observations in the other clinical trials for ABT-773. The other clinical trials for ABT-773, which had included several hundred patients, had not demonstrated clinically significant liver toxicity issues. After analysis of the results of this study we decided to repeat it, since we believed that the subjects selected for the study as well as some methodological issues skewed the results of the trial. The repeated study, which was completed in late 2000, demonstrated that the liver toxicity issues observed during the first study were not reproduced, and we concluded that the findings in the first Japanese phase I trial were not reliable and unlikely to be related to ABT-773. Attached hereto as D's Exhibit 587 is a true and correct copy of a January 2001 Monthly Project Status Report for ABT-773 that I received in January or February 2001. As reflected in the document, we had concluded in January 2001 that

> ***ABT-773 is clear in terms of hepatotoxicity profile*** and the liver enzyme profile abnormality observed in Hawaiian Ph I with Japanese population was seen as a result of the high fat diet during the study period.
> *Id.* at 0000302 (emphasis added).

75. None of the information presented in the February and March 2001 PEC updates on ABT-773 or the March Portfolio Review Presentation for ABT-773 led me to believe that ABT-773 had any hepatotoxicity issues. As reflected in those presentations, there was no evidence of liver function test increases in Japanese or Caucasian patients after the repeated Phase I study. D's Exhibit 607 at ABBT0576846; D's Exhibit 622 at ABBT0013212; D's Exhibit 631 at ABBT120481.UR.

76. As of March 2001, we did not have any clinical data that indicated there were any clinically significant liver issues with ABT-773. Nor did anyone on the ABT-773 development team inform me that they believed there were any such issues. In late 2001, the ABT-773 development team began a Phase I clinical trial to evaluate potential QT prolongation, which was referred to as the M01-325 trial. Since the FDA was, at this time, working on formal guidance for the industry regarding specific ways to measure the potential for QT prolongation issues, we decided to put our own policy in place that would include additional trials, such as this one, specifically focused on the issue of QT prolongation. ABT-773 was one of the first drugs to be subjected to this policy. During that trial, there were unexpected liver function test elevations seen in four patients. Until that point, I had not seen any credible evidence that there was a potential liver toxicity issue with ABT-773. Attached hereto as D's Exhibit 756 is a true and correct copy of the Monthly Highlights Memorandum from November 9, 2001 that I circulated reflecting the liver function test elevations during that clinical trial.

The Dosing of ABT-773

77. As of March 2001, we had determined that ABT-773 would be developed for once-a-day dosing for the two less severe indications for which it was being developed, chronic bronchitis and pharyngitis. It was unclear, however, as of March 2001, whether the two more severe indications, community-acquired pneumonia ("CAP") and acute bacterial or maxillary sinusitis, would be dosed at once-a-day, commonly referred to as QD dosing, or twice-a-day, commonly referred to as BID dosing. We anticipated making the decision regarding the dosing of the two more severe indications in the summer of 2001 after we had obtained Phase III clinical trial results that would aid

in the decision-making process. Attached hereto as D's Exhibit DN is a copy of the April 2001 Monthly Highlights Memorandum that I circulated reflecting that the dosing decision for ABT-773 was to be made during the summer of 2001. On July 25, 2001, a decision analysis regarding the dosing of ABT-773 was presented to Dr. Leiden and me by the ABT-773 development team. Attached as hereto as D's Exhibit DO a true and correct copy is the Monthly Highlights Memorandum from August 10, 2001 that reflects the outcome of that meeting. Based on the information presented during the meeting, we decided to pursue BID dosing for the more severe indications, community-acquired pneumonia and acute bacterial or maxillary sinusitis. Our decision was based on the fact that we had insufficient information, as of July 2001, to determine whether QD dosing would be considered sufficient by the FDA for those indications. The choice we made was to go ahead with BID dosing for the more severe indications in order to keep the development of the compound on track. Additionally, we realized that if we were later able to satisfy the FDA that QD dosing was appropriate for the more severe indications, we could introduce such dosing post-launch, thus minimizing any potential negative commercial impact that might result from an initial BID launch for the more severe indications.

The Pediatric Program

78. We always intended to develop a pediatric formulation for ABT-773, if possible, and the ABT-773 program had a plan to accomplish that goal from 2000 on. As noted in the December 2000 presentation on ABT-773 that I attended, we initiated the pediatric program for ABT-773 in January 2000. D's Exhibit 787 at ABBT205238. In September 2000, the ABT-773 development team conducted its first taste evaluations of

the oral formulation of the compound intended for pediatric use and found that it had a bitter taste that would place it at a competitive disadvantage; it therefore needed to be reformulated. As set forth in the December 2000 presentation, the ABT-773 team had developed a pediatric program plan that contemplated the development of a new pediatric formulation with a Go/No Go decision in June 2001, and which also contemplated clinical testing of that pediatric formulation. Since the ABT-773 team was principally focused on the tablet formulation, we decided to delay the development of the pediatric formulation until the tablet formulation Phase III clinical trials were underway.

79. Pharmaceutical companies usually develop and study the pediatric patient populations with a new drug only after the corresponding adult program has acquired a significant amount of adult data. It is generally judged unacceptable to expose children to products without having demonstrated substantial activity and especially safety in adults first. The FDA's Pediatric Rule did not require that we develop a pediatric formulation for ABT-773 before the adult tablet could be launched. The Pediatric Rule only required Abbott to initiate pediatric work at some time prior to the regulatory approval of the adult formulation. Moreover, the Pediatric Rule also contemplates that a drug sponsor can obtain a waiver or a deferral of the requirements of the rule under certain circumstances.

80. On September 18, 2001, I sent an email to Dr. Bukofzer and Ms. Meyer asking about the status of the pediatric program for ABT-773. Attached hereto as D's Exhibit AL is a true and correct copy of my September 18, 2001 email and Dr. Bukofzer's responses to the questions posed in that email. As noted in Dr. Bukofzer's response, work on a new formulation for the pediatric program was expected to begin in

October 2001 and the first clinical trial for the new formulation was expected to start six months later. *Id.* at ABBT203480. In fact, formulation work for the Pediatric Program began in October 2001. Attached hereto as D's Exhibit DH is a true and correct copy of the October 8, 2001 Monthly Highlights Memorandum that I prepared reflecting that additional formulation work was being undertaken in October 2001.

81. During 2000 and 2001, I did not believe that the FDA's Pediatric Rule would pose an obstacle to our ability to obtain regulatory approval from the FDA to launch ABT-773. Nor did I believe that the requirements of the rule would delay that launch. I believed that the work we had done and intended to do on the ABT-773 pediatric program would be sufficient to satisfy the FDA that we had met the requirements of the rule, or that we could obtain a waiver or deferral of those requirements.

The April 2001 Ketek Advisory

82. In April 2001, the Anti-Infective Drugs Advisory Committee for the FDA held its first Advisory Committee meeting for Ketek, a ketolide that was under development by Aventis, another pharmaceutical company. An FDA advisory committee is a group of outside experts who provide advice to the FDA regarding specific areas under the purview of the FDA. The opinions expressed by the FDA's advisory committees carry significant weight in the FDA's determination of whether a drug is approved or not, and frequently the Advisory Committee's deliberations will determine the nature of additional clinical investigation before or after approval is granted.

83. As of early 2001, Ketek was at a more advanced stage of development than any other ketolide. We had expected that the Advisory Committee would be

focused principally on efficacy concerns since there were so many efficacious anti-infectives already on the market. In fact, however, the Advisory Committee focused very heavily on the size of Ketek's safety database for both QT prolongation and liver toxicity. We had expected to run a number of Phase III and additional Phase I trials for ABT-773, with the understanding that we would need to demonstrate that ABT-773 was clear of QT prolongation and liver toxicity issues. Based on only a small number of incidents of elevated liver function tests in the Ketek clinical trials, the FDA was requiring additional clinical trials that included over 20,000 patients. Based on the four incidents of elevated liver function tests that we observed in the October 2001 Phase I trial, we were concerned that we would also be required to conduct additional clinical trials that would include 20,000 patients as well.

84. The Advisory Committee meeting demonstrated to us that the safety hurdle for anti-infectives, and with that ketolide anti-infectives, had increased well beyond our original understanding. The evolving standard to demonstrate the absence of an issue in far larger numbers of patients than what was traditionally the case for anti-infective programs meant that the expense and likely duration of developing the compound as well as adequately demonstrating its safety would be substantially increased. Based on the Ketek advisory, we realized that the expected value of ABT-773 had fallen dramatically. The Ketek advisory revealed that Aventis would be required to perform a greater than 20,000 patient study to determine the incidence of liver toxicity because of only two specific cases of liver toxicity that had occurred in the Ketek database. This information was significant because up until that time our experience was that a very low incidence of liver toxicity in a clinical trial did not affect the FDA's

recommended safety database so greatly. As noted above, in October 2001, we had observed convincing evidence of elevated liver function tests in one of our ABT-773 studies in October 2001. We realized that the newly expanded study was expected to cost Aventis over $100 million and last several years, a cost that we had not accounted for in the development of ABT-773. In sum, the information we received after the Ketek advisory made it clear that the cost of developing ABT-773 had increased substantially from what we had initially planned.

The Discontinuation of ABT-773

85. In late 2001, I attended a PEC up-date presentation regarding ABT-773 that was given by Dr. Bukofzer. Attached hereto as D's Exhibit 760 is a true and correct copy of the Summary of the December 1, 2001 PEC meeting that I received after that meeting. Also attached as D's Exhibit EC is a true and correct copy of the presentation given by Dr. Bukofzer at that meeting based on the ABT-773 clinical data that had been acquired and analyzed by late 2001. Based on the results and implications of the Ketek Advisory Committee's findings, the PEC made the decision not to start any new ABT-773 activities or studies, but to continue all ongoing activities and studies.

86. On January 9, 2002, I attended a meeting with Mr. Miles White, Abbott's Chief Executive Officer and Chairman of the Board**,** regarding ABT-773. Attached hereto as D's Exhibit DQ is a true and correct copy of the meeting notice for that meeting. Prior to that meeting, Dr. Bukofzer and Dr. Sun drafted and circulated a Memorandum, dated January 22, 2002, concerning the development status of ABT-773. Attached hereto as D's Exhibit 761 is a true and correct copy of that Memorandum, which I received on or around January 17, 2002. I believe that Dr. Bukofzer, Dr. Sun,

Dr. Leiden and Mr. Arthur Higgins also attended this meeting with Mr. White. At this meeting, we discussed generally the fact that information had been developed since April 2001 that indicated that ABT-773 had significantly deviated from its target product profile. I believe the January 2002 Memorandum accurately reflects the details of what was discussed during that meeting.

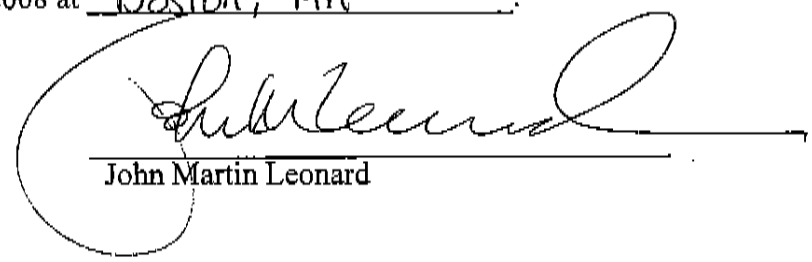
87. At the end of 2001 and the beginning of 2002, we found ourselves increasingly in the position of trying to prove a negative with regard to ABT-773. While we did not believe that ABT-773 had any specific QT prolongation or liver issues that were worse than other anti-infectives that had been approved by the FDA and that had already been marketed successfully, the Ketek advisory in April demonstrated to us that the FDA would require substantial additional work and patients than we had originally forecast for the program. At that time we realized that the program could be far longer and much more expensive than we had originally intended it to be.

88. Work on ABT-773 projects that had been initiated prior to the PEC meeting continued through the beginning of 2002. Attached hereto as D's Exhibit DR is a true and correct copy of the February 8, 2002 Monthly Highlights Memorandum that I circulated that reflects that the Phase I QT study amendment had been approved and that the study was scheduled to re-start by the end of February 2002. Additionally, two ongoing studies were enrolling additional patients during January 2002. A Phase I study was re-started in March 2002 and there was one additional study that was ongoing in March 2002. Attached hereto as D's Exhibit DS hereto is a true and correct copy of the April 9, 2002 Monthly Highlights Memorandum I circulated reflecting the ongoing activities for ABT-773.

89. Eventually, in the summer of 2002, after careful consideration, the PEC made the decision to discontinue the development of ABT-773 and to out-license the compound based on the new information discussed above regarding challenges to the development of ABT-773 that had become available after April 2001, including the clinical data accumulated since that date that indicated that ABT-773 was deviating significantly from its target profile and the information learned as a result of the Ketek advisory.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Executed on March 8, 2008 at Boston, MA.



John Martin Leonard